centrations and to favorably affect outcome. Charcoal should be used in virtually all overdose cases; most tested chemicals and drugs are adsorbed, and there are few contraindications. Charcoal should be avoided if it adsorbs an orally given antidote used in a specific poisoning. Adsorption of orally administered acetylcysteine by charcoal has been suggested in one study, but was seemingly absent in another. Until this is clarified activated charcoal should probably be withheld in major acetaminophen overdoses. Likewise, charcoal will adsorb orally administered ethanol, and thus should be withheld in methanol and ethylene glycol overdose.

Charcoal palatability can be increased by the addition of sorbitol or saccharin, the latter of which can be used in a 1:20 dilution and thus not increase total volume of charcoal that has to be swallowed.

Cathartics have not been proved to be beneficial in overdose cases, but recent studies show that at least in some cases they actually increase the adsorptive capacity of charcoal. Magnesium cathartics, in particular, increase cathartic removal of salicylates and several other substances.

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Coelenterate (Man-of-War) Envenomation

RECREATIONAL AND INDUSTRIAL PURSUITS in coastal tropical waters have increased the number of envenomations inflicted by coelenterates (cnidarians) on humans. The most frequent offenders are *Physalia physalis,* the Atlantic Portuguese man-of-war, and *Physalia utriculus* (bluebottle), the Pacific version. These predators are free-swimming, pelagic organisms composed of a nitrogen and carbon monoxide-filled sail (pneumatophore) up to 30 cm long from which are suspended nematocyst (venom organelle)-laden tentacles. These tentacles may be numerous and measure up to 30 m (100 ft) in length *(P physalis).* When the animal moves in the ocean, these structures coil and fold to produce stinging "batteries," which may involve more than a million nematocyts.

Nematocyst venom includes toxic fractions that can invoke any and all of anaphylaxis, muscle spasm, exquisite pain and neurologic-cardiovascular collapse. Venom components include adenosinetriphosphatase, fibrinolysin, hyaluronidase, histamine, peripheral calcium antagonists, myocardial depressants, hemolysins and dermatonecrotic agents. Milder envenomation may provoke only an irritant dermatitis, whereas severe stings may induce generalized multisystem failure.

The immediate therapy for coelenterate stings is gentle topical application of isopropyl alcohol (40 percent) or acetic acid (5 percent). Fresh water or abrasion will discharge unexploded nematocysts and should be avoided. Following the initial detoxification, the remaining organelles can be removed by shaving the affected area. All patients should receive appropriate tetanus prophylaxis. Muscle spasm and pain are controlled with the administration of calcium gluconate and narcotics, respectively. Severe envenomations may require advanced life support. Washed-up tentacle fragments can retain activity for months. In children who ingest these, acute airway obstruction may develop from local oropharyngeal edema. Prompt detoxification and airway management is often lifesaving. Steroid administation has not been shown to be of definitive benefit.

There is not yet an effective antivenin for the sting of *Physalia,* as there is for *Chironex fleckeri* (box-jelly or sea wasp). The increased incidence of envenomations has provided impetus for a growing interest in the development of such an agent.

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Mechanism of Blood Flow During Cardiopulmonary Resuscitation

CARDIOPULMONARY RESUSCITATION (CPR) is a therapeutic method that over the past 20 years has proved to be the critical first component in the management of sudden cardiac death. New concepts and controversies have recently been generated regarding the mechanism of blood flow during CPR: The central issue and major controversy is whether blood flow results from compression of the heart between the sternum and spine, as initially espoused by Kouwenhoven and colleagues, or whether antegrade flow is primarily due to the generalized rise in intrathoracic pressure that occurs during the act of chest compression, as suggested by animal and human investigations at Johns Hopkins University, the University of California, the Baylor College of Medicine and the University of Washington.

According to the latter theory, blood flow during CPR results from rhythmic increases in intrathoracic pressure during chest compression. The increase in intrathoracic pressure is transmitted directly to the extrathoracic arterial bed but, due to closure of venous valves, not to the peripheral venous system. A peripheral arterial-to-venous perfusion gradient is thus established that facilitates blood flow. This theory is supported by the finding that pressures in the cardiac chambers and ascending aorta rise to a level equal to the change in intrapleural pressure during chest compression. (If selective cardiac compression were to occur, ventricular pressures would exceed atrial pressures). In addition, cineangiographic studies in animals and two-dimensional echocardiographic studies in hu-

mans have shown that during CPR the left heart functions more as a passive conduit than as a pump. Implicit in the "chest pump" hypothesis for blood flow is that manipulations that increase intrathoracic pressure during CPR increase left heart outflow.

Regional perfusion studies using radiolabeled microspheres have shown a significant improvement in cerebral flow when simultaneous chest compression and lung inflation are used during CPR. However, simultaneous chest compression and lung inflation do not improve myocardial perfusion. In fact, coronary flow using conventional or newer methods of CPR averages only 1 percent to 3 percent of control flow!

Complications of newer CPR techniques in studies using animals appear to be minimal. Barotrauma from high airway pressures is infrequently noted and ventilation is more than adequate (provided a patient is intubated and positive pressure is used). The efficacy and safety of simultaneous chest compression and lung inflation in improving survival for victims of sudden death is being assessed in a Miami field study.

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Compelling Treatment After Suicide Attempts

A LARGE PROPORTION of the poisonings seen in emergency departments are actually the result of suicide attempts or suicide gestures. Patients frequently ingest a potentially lethal dose of a drug and then call for help. This seemingly paradoxical behavior may continue in the emergency department when the patient readily admits to a drug ingestion but refuses treatment. The attending physician is presented with an apparent legal and ethical dilemma: a patient who needs help yet refuses therapy.

The fundamental legal principle that people have the right to make major decisions about their bodies has long been recognized by the courts. In a 1914 decision Cardozo noted, "Every human being of adult years and sound mind has a right to determine what shall be done with his own body; and a surgeon who performs an operation without his patient's consent commits an assault for which he is liable in damages."

Some authors cite the 1960 case of *Natanson v Kline* as the first suit in the modern era of malpractice of a physician who failed to receive informed consent for treatment. During the 20 years since *Natanson,* the doctrine of informed consent has had a rapid evolution. Clearly a competent adult has a legal right to refuse treatment. Numerous cases show that this right extends to the refusal of potentially lifesaving therapy, except when another person or the state has a compelling interest in the patient's continued life. Legislation to permit persons to direct the conditions for their own terminal care has become commonplace, with nearly 100 different acts proposed or enacted in the United States by 1978.

Patients' rights notwithstanding, a drug overdose patient must be treated in an emergency department. Suicide has been defined as the intentional, voluntary, nonaccidental taking of one's own life. Where it has been shown that a person's refusal to submit to medical treatment is likely to result in death, which death may be classified as a suicide, the state may compel treatment. It has been argued that accepting a patient's refusal of treatment for a suicide attempt may be aiding in the suicide, thus leaving a physician criminally liable. The Lanterman-Petris-Short Act directs that any person who "is a danger to others or to himself, or gravely disabled" shall be placed in an approved "facility for 72-hour treatment and evaluation." Under the act, " 'Intensive treatment' consists of such hospital and other services as may be indicated."

We advise emergency physicians to obtain a signed informed consent when possible. However, all conditions that are an immediate threat to life or limb should be treated regardless of ability to obtain consent. This includes treating potentially life-threatening poisonings and overdoses. A psychiatric evaluation is a mandatory part of emergency treatment of drug overdose. The evaluation should be done as early as medically possible, and certainly before a patient is permitted to leave the emergency department. After a patient is in a stable condition and out of immediate danger from a delay in treatment, the physician should contact a probate court for directions on further, less urgent therapy.

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Overdose Update—Antidotes

SPECIFIC ANTIDOTES are unnecessary in almost all cases of overdose, but can be lifesaving in a few selected instances. A number of recently described or experimentally promising antidotes deserve mention.

Treatment with the oral administration of acetylcysteine in the United States, or intravenously given acetylcysteine and orally given methionine in Great Britain, has been shown to greatly diminish the incidence of significant hepatic necrosis following acetaminophen overdose. Effects on renal toxicity are not known. Patients found to have blood concentrations suggesting possible toxicity on standard acetaminophen nomography studies (which must be evaluated with regard to amount of time following drug ingestion) should be begun on a standard protocol of 18 doses of acetylcysteine.

Alkalinization of the serum to a pH of 7.50 to 7.55